Eosinophilic gastroenteritis in a dog

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Abstract — A 9-year-old mixed-breed dog was evaluated for chronic intermittent vomiting, hematemesis, and melena lasting several months. Biopsy specimens obtained during exploratory laparotomy revealed eosinophilic gastroenteritis. Treatment included drug therapy to reduce gastrointestinal inflammation and dietary management to limit antigenic exposure.

Résumé — Gastro-entérite éosinophile chez un chien. Un chien croisé âgé de 9 ans a été évalué suite à des vomissements chroniques intermittents, de l'hématémèse et du méléna qui duraient depuis plusieurs mois. Des biopsies prélevées au cours d'une laparotomie exploratoire ont révélé une gastro-entérite éosinophile. Le traitement comprenait une thérapie médicamenteuse visant à réduire l'inflammation gastro-intestinale et des mesures diététiques afin de limiter l'exposition antigénique.

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A 9-year-old, 7-kg, neutered male, mixed-breed dog was presented with a history of intermittent vomiting lasting 1 wk. Although initially the vomitus had contained clear fluid, more recently frank blood was reported. When examined on day 1, the dog appeared bright and responsive, with no evidence of either diarrhea or dehydration. The dog was mildly pained during abdominal palpation, but no other signs of abnormalities were detected. Lateral and ventrodorsal survey and contrast abdominal radiographs revealed a thickened gastric wall and a small amount of gas in the small intestine. A complete blood cell (CBC) count and blood biochemical profile were declined by the owner.

A tentative diagnosis of uncomplicated gastroenteritis was made, and symptomatic treatment was prescribed for 5 d as follows: metoclopramide (Apo-Metoclop; Apotex, Toronto, Ontario), 0.35 mg/kg bodyweight (BW), PO, q8h; cimetidine (Apo-Cimetidine; Apotex), 7.1 mg/kg BW, PO, q8h; and sucralfate (Sulcrate Suspension Plus; Hoechst Marion Roussel, Laval, Quebec), 0.35 mg/kg BW, PO, q8h. The dog improved initially, but began vomiting clear fluid 2 wk after therapy ended (day 20). Metoclopramide, 0.35 mg/kg BW, PO, q8h, was dispensed for a further 3 d, and a bland diet (Medi-cal Gastro; Veterinary Medical Diets, Guelph, Ontario) was recommended.

The dog showed improvement after this course of therapy, but hematemesis and melena were evident 2 wk later (day 36). At that time, physical examination was unremarkable. A CBC count (QBC VetAutoread Hematology System; IDEXX Laboratories, Westbrook, Maine, USA) and biochemical profile (VetTest Chemistry Analyzer; IDEXX Laboratories) revealed no abnormalities. Differential diagnoses included gastric or intestinal neoplasia, gastric ulceration, inflammatory bowel disease, and small intestinal bacterial overgrowth. The owner elected to continue with symptomatic therapy, which included cimetidine, 7.1 mg/kg BW, PO, q8h; sucralfate, 0.35 mg/kg BW, PO, q8h; metronidazole (Novo-Nidazol; Novopharm, Toronto, Ontario), 9 mg/kg BW, PO, q8h; and amoxicillin (Amoxil; Wyeth-Ayerst, Montreal, Quebec), 14 mg/kg BW, PO, q8h. A bland diet (Medi-cal Gastro; Veterinary Medical Diets) was again prescribed.

Clinical signs resolved for a further 10 wk (day 109), when the dog was examined again because of hematemesis. The stools appeared normal at the time of examination, and a fecal flotation was negative for internal parasites. The dog, which had lost 0.6 kg BW since the initial visit, appeared thin, but no other abnormalities were detected. Lateral and ventrodorsal abdominal radiographs again suggested a thickened gastric wall. The owners declined a referral for gastric endoscopy, or ultrasound, or both, but did opt for an exploratory laparotomy and gastrotomy.

Few gross abnormalities were visualized during surgery. The duodenum was slightly thickened on palpation but was normal in appearance. There were a few small areas of serosal hyperemia in the ileum. Full thickness intestinal biopsies were taken from the proximal part of the duodenum and distal part of the ileum, and mucosal biopsy samples were taken from the gastric mucosa via gastrotomy. All samples were placed in 10% buffered formalin. Histological evaluation revealed extensive infiltration of the submucosa and lamina propria by eosinophils, lymphocytes, and plasma cells. A

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diagnosis of severe eosinophilic gastroenteritis (EGE), a form of inflammatory bowel disease (IBD), was made on the basis of the dog's clinical signs and the histopathological findings.

Treatment was initiated with prednisone (Apo-Prednisone; Apotex), 1 mg/kg, BW, PO, q24h for 14 d, to be gradually decreased to 0.35 mg/kg BW, PO, q48h, over 2 mo. A new diet (Medi-cal Vegetarian Diet; Veterinary Medical Diets) was introduced, and metronidazole, 35 mg/kg BW, PO, q12h for 1 mo, was also prescribed.

Eight weeks after initiation of therapy (day 168), the dog was reported to be doing well, without further evidence of vomiting or melena. The dosage of prednisone was tapered to 0.35 mg/kg BW, q48h, with the hopes of maintaining the dog on dietary therapy alone, if the clinical response continued to be favorable.

Although IBD is the most common cause of chronic vomiting and diarrhea in the dog, diagnosis may be difficult (4). The history usually includes chronic or intermittent vomiting, diarrhea, inappetence, and weight loss. Hematemesis, hematochezia, or melena may be present as a result of gastrointestinal ulceration, particularly with EGE (1,2). As in this case, results of physical examination may be unremarkable, but there may be thickened loops of bowel (1,2) or mesenteric lymphadenopathy (2). Radiographic findings are usually normal, although contrast studies may reveal mucosal irregularities or thickening of the intestinal or gastric wall, as in this case (2). Peripheral eosinophilia may occur with EGE, although this is not a consistent finding (1–3), and was not evident in this case. Regenerative anemia may be associated with gastrointestinal ulceration (2). In this dog, hematemesis and melena occurred without anemia. Panhypoproteinemia may be found in dogs with EGE, reflecting protein-losing enteropathy due to diffuse small intestinal involvement (2).

Ultimately, IBD is a diagnosis of exclusion. Other causes of intestinal inflammation, including infection with *Giardia* spp., other intestinal parasites, or pathogenic bacteria, must be eliminated before invasive diagnostic procedures are carried out (1,4). Biopsy samples may be collected for historical analysis, either by endoscopy or at laparotomy. Since inflammatory cells are normally present in gastric and intestinal tissues, it is important that the histopathologist be experienced in grading IBD lesions (1,3). Histologically, IBD is characterized by infiltrates of inflammatory cells in one or more segments of the gastrointestinal tract (1,2,4). The predominant cell type dictates the type of IBD, and the nomenclature is dependent upon the region of the tract that is affected. Lymphocytic-plasmacytic enterocolitis (LPE), the most common type of IBD in dogs, is characterized by large numbers of lymphocytes, plasma cells, and other inflammatory cells within the lamina propria of the small and large intestine (3,4). The second most frequent form of IBD is EGE, which is typified by an infiltrate of eosinophils, often with lesser numbers of other inflammatory cells (such as lymphocytes and plasma cells) in the mucosa and submucosa of the stomach and small intestine (1–3).

The etiopathogenesis of IBD is unknown. Many researchers agree that hypersensitivity of the gut-associated lymphoid tissue (GALT) to antigens in the gastrointestinal tract is involved (3). Whether this hypersensitivity is a result of a primary defect in the immunoregulation of GALT, or is secondary to inflammation caused by an unidentified pathogen, has not been established (1,3). Changing to a hypoallergenic diet may help to alleviate clinical signs in some dogs, supporting a role for dietary antigens in the pathogenesis of EGE (1). Regardless of the inciting cause(s), an inflammatory cascade ensues, with the release of chemical mediators that cause tissue damage and the release of other chemotactic factors that attract more inflammatory cells (1).

Since the cause(s) of IBD have not been established, treatment of the disease focuses on control of clinical signs. Mild IBD may respond to a change in diet, to a hypoallergenic or elimination formula, without drug therapy (1). In severe cases, dietary therapy may alleviate the need for high doses of drugs to achieve control of clinical signs (1). However, as in the case reported here, most patients require both dietary management and pharmacological treatment (1).

Dogs with IBD may be malnourished and have increased needs for protein and energy (3). The diet must be highly digestible and hypoallergenic; it must also be palatable and provide the energy and nutrients required. Many dogs with IBD respond favorably to an elimination diet, which should have a single, novel protein source and a highly digestible, gluten-free carbohydrate source (1,3,4). A low fat diet is more easily tolerated, as it is less likely to delay gastric emptying (4). If the colon is affected (EGE), increasing dietary fiber may alleviate signs of large bowel involvement (diarrhea and tenesmus) (1). Fiber increases fecal bulk, improves colonic motility, and binds water and irritants (1,4). Many hypoallergenic formulations are now commercially available. Although homemade recipes are available, they may be unsuitable for long-term feeding (3,4). Owners must be made aware that treats, table scraps, flavored medications, and rawhides may contain antigenic components and should be avoided (3,4). Dogs with severe or refractory IBD may benefit from commercially available formulations containing starch and a hydrolyzed protein source (Ralston Purina CNM HA-Formula; Ralston Purina, St. Louis, Missouri, USA). These protein and carbohydrate sources are presented as smaller molecules and may be less likely to evoke a hypersensitivity response (4,5).

Soon after treatment for IBD is initiated, inflammation is reduced and mucosal permeability to dietary antigens increases. As a consequence, some patients may become hypersensitive to the novel protein source in the hypoallergenic diet, ultimately delaying recovery (1,3,4). Introducing a different novel protein 6 wk after beginning treatment, when inflammation has been controlled, is recommended (1,3,4).

Most patients require some form of pharmacological therapy as an adjunct to dietary control (1,4). Corticosteroids are the most common pharmaceutical prescribed for IBD in dogs, providing both immunosuppressive and anti-inflammatory effects (4). Oral corticosteroids are commonly administered at an induction dose of 1 to 2 mg/kg BW, q12h, gradually tapered every 1 to 2 wk

(1). New corticosteroid products that are eliminated primarily by first-pass hepatic metabolism are currently being investigated for use in dogs with IBD. Budesonide, an agent used for many years in humans with IBD, acts topically within the gastrointestinal tract, so that many of the side effects commonly associated with systemic use of corticosteroids are avoided (6).

Other agents may be used either alone or in combination with corticosteroids. In severe or refractory cases, azathioprine may be useful to maintain remission or decrease the use of corticosteroids. This drug may cause bone marrow suppression, so periodic hematological monitoring is necessary (4). Metronidazole, often used to treat canine IBD, has several beneficial effects. It inhibits cell-mediated immunity, is antiprotozoal, and is bactericidal against anaerobic bacteria (4). Sulfasalazine is useful in cases that involve the large bowel. The metabolites of this compound inhibit inflammatory mediators within the colonic mucosa (1). Careful monitoring of tear production is recommended during treatment with sulfasalazine, as keratoconjunctivitis sicca is one of the more common side effects (1).

After 2 or 3 mo of therapy, many dogs can be weaned off all medication, while remaining on a controlled diet (1). Since treatment is aimed at control of the clinical signs, rather than a cure, relapses are common and should be expected (1). It is important to educate owners about the importance of following dietary recommendations, as most relapses are associated with dietary indiscretion (1).

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References

1. Jergens AE. Inflammatory bowel disease: Current perspectives. Vet Clin North Am Small Anim Pract 1999;29:501–521.
2. Johnson SE. Canine eosinophilic gastroenterocolitis. Semin Vet Med Surg 1992;7:145–152.
3. Guilford WG. Idiopathic inflammatory bowel disease. In: Guilford WG, Center SA, Strombeck DR, Williams DA, Meyer DJ, eds. Strombeck's Small Animal Gastroenterology. 3rd ed. Philadelphia: WB Saunders, 1996:451–486.
4. Marks SL. Management of canine inflammatory bowel disease. Compend Contin Educ Pract Vet 1998;20:317–331.
5. Davenport DJ, Remillard RL, Simpson KW, Pidgeon GL. Gastrointestinal and exocrine pancreatic disease. In: Hand MS, Thatcher CD, Remillard RL, Roudebush P, eds. Small Animal Clinical Nutrition. 4th ed. Marceline: Walsworth Publ, 2000: 725–810.
6. Stewart A, Bolineck J. The use of a novel formulation of budesonide as an improved treatment over prednisone for inflammatory bowel disease. J Vet Intern Med 1997;11:115.